trophin-releasing hormone (TRH). Seven patients showed evidence of primary pituitary deficiency, five of whom showed low thyroid function, while the basal serum TSH level was not elevated and failed to rise after TRH administration. Thus damage to both the normal hypothalamus and the normal pituitary gland may occur following irradiation in the dose range 5000-8300 rads.

Further points in the article concern the effects on GH production of neurosurgery and radiation to parts of the brain other than the hypothalamic-pituitary region. We have studied more than 30 children after neurosurgery and before irradiation. No child has shown impaired GH responses to provocative stimuli before irradiation.[3] Furthermore no child whose hypothalamic-pituitary axis totally avoided irradiation has ever shown inadequate GH responses. Much more interesting is the question whether or not irradiation of other parts of the brain may interfere with somatic growth by a non-GH-mediated mechanism. There is some evidence that this occurs in the rat.[4-6]

Finally, the question is raised at the end of your article about possible growth responses to exogenous GH. We have six children with radiation-induced GH deficiency on GH therapy. The mean growth velocity during the pretreatment year was 3·7 cm and during the first year of GH therapy 7·9 cm. These data have not yet been published,[3] but other authors have described similar increases in growth velocity with GH therapy in such children.[7 8]

It is always pleasing to see a leading article in the *BMJ* related to a topic one is particularly interested in but disheartening when one finds the article so ill-informed as this one.

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[6] Mosier, H D, and Jansons, R A, *Radiation Research*, 1970, **43**, 92.
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****We are glad of the opportunity to apologise to Dr Shalet and his colleagues for missing their "short communication,"[1] which appeared some time after the paper we discussed and which went some way to remedying its main defect, but in our view the first paper should not have been published without this dosage information (which was presumably readily available) or at the very least without a note to show that the authors appreciated its importance and would be giving the relevant data in a subsequent communication. Unfortunately, there is a further serious omission in this second communication. Nothing is said about why some patients were given a higher dose of radiation than others. No matter how impressive the statistical significance of the inverse correlation found between radiation dose and peak GH response, the possibility that those patients given a higher dose were in some important way different from those given a lower dose ought to have been looked at and commented on. We look forward to clarification of this point in the unpublished paper mentioned in their letter.

With regard to the normal pituitary gland being "unlikely to be affected by quite high doses of radiation" we feel that the context and the references we gave made it clear that we were referring to the fact that long-term follow-up of large numbers of adult patients has provided no evidence of any *clinical* hypopituitarism in the great majority. This point has again been emphasised recently by Bloom.[2] It is important that the risks of radiation should be neither minimised nor exaggerated.—ED, *BMJ*.

[1] Shalet, S M, *et al*, *Clinical Endocrinology*, 1976, **5**, 287.
[2] Bloom, H J G, *Proceedings of the Royal Society of Medicine*, 1977, **70**, 319.

Danger of salt as an emetic

SIR,—Dr N C Hypher's recent testimonial (16 April, p 1033) championing the use of salt as an emetic is most unfortunate indeed. Your own pages carried the report of a fatality consequent to such use 14 years ago[1] and again three years ago.[2 3] In addition our group and at least three others have documented the danger of such an approach with enormously elevated serium sodium concentrations (for example, 214 mmol(mEq)/l) and death as a direct consequence.[4-7] Making some conventional assumptions just two tablespoons of salt could lead to a 30 mmol(mEq)/l increase in serum sodium.[8] Moreover, in the United States the Consumer Product Safety Commission (*Federal Register*, 23 June 1977) has proposed a policy calling for the elimination of this archaic and dangerous approach in all first-aid labelling practices and the substitution of syrup of ipecacuanha as *the* emetic agent of choice. While far from ideal, it is reasonably effective, easily obtainable, and, in the syrup form, totally devoid of any serious toxicity despite its almost routine use in poison centres throughout the United States for more than 10 years. Salt certainly has its place—but not as an emetic. Any recommendation for such use—as a first-aid measure or otherwise—ought to be restricted from publication, incurring as it would potential liability for both author and publisher.

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[2] Winter, M, and Taylor, D J E, *British Medical Journal*, 1974, **3**, 802.
[3] Bird, A, *British Medical Journal*, 1974, **4**, 103.
[4] Robertson, W O, *Journal of Pediatrics*, 1971, **79**, 877.
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[6] Barer, J, *et al*, *American Journal of Diseases of Children*, 1973, **125**, 889.
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Cimetidine and gastric carcinoma

SIR,—Cimetidine is now well established as effective in the treatment of duodenal ulceration[1] and the recent report by Dr F Frost and others (24 September, p 795) suggests that it is almost as effective in promoting the healing of gastric ulcers.

While patients with peptic ulcers will undoubtedly benefit from this advance, there is a possibility that as this form of treatment becomes more widespread patients with early gastric carcinoma who present with the same symptoms as those with benign peptic ulceration will be overlooked. It is well established that malignant gastric ulcers will heal and often become symptom-free on careful conservative management, although the cancer continues to proliferate within the stomach wall. If dyspeptic patients can have their symptoms relieved with cimetidine the incidence of missed gastric carcinomas, which is already unacceptably high, will rise even higher.

In a recent survey of gastric carcinoma treated at this hospital over the past 13 years only 43% of patients were operable and of these one-third had palliative resections. The earliest presenting symptom in this series was dyspepsia, which had been present for an average of five months by the time the patient was referred to the hospital, and many patients had been receiving antacid medication from their family practitioner for some months.

Patients over the age of 50 with persistent dyspepsia for two months or more should be fully investigated with a barium meal and gastroscopy before conservative treatment is continued.

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[1] Gray, G R, *et al*, *Lancet*, 1977, **1**, 4.

Perforation of peptic ulcer after withdrawal of cimetidine

SIR,—I am surprised that Mr W A Wallace and his colleagues (1 October, p 865) have implicated cimetidine in perforation of peptic ulcers. They cite no further evidence than that three patients out of 17 who perforated had previously stopped cimetidine abruptly. In view of the popularity of this new drug it seems hardly surprising that some of the patients should have been on cimetidine and that three should have stopped it suddenly. Most patients stop drugs suddenly. This is hardly proof of a causal relationship and on this basis to suggest continuing maintenance for three months irrespective of symptoms seems wrong.

Further, to castigate their general practitioners for treating without immediate prior investigation is unrealistic. Two of the patients had had previous barium meals showing ulceration and I do not consider that prescribing cimetidine is, on its own, sufficient reason for reinvestigation as this would deny its benefit to many patients owing to the clogging-up of the various hospital departments for reinvestigation.

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SIR,—Dr W A Wallace and others (1 October, p 865) report three cases of peptic ulcer perforation in patients who had undergone recent treatment with cimetidine. They suggest that abrupt cessation of treatment may have precipitated the perforations. I append details of a similar case encountered in this general practice.